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=> s chemokine (s) peptide (s) inhibitor

L1          202 CHEMOKINE (S) PEPTIDE (S) INHIBITOR

=> s chemokine (s) peptide (s) inhibitor (s) mcp

L2           36 CHEMOKINE (S) PEPTIDE (S) INHIBITOR (S) MCP

=> dup rem l2

PROCESSING COMPLETED FOR L2
L3           19 DUP REM L2 (17 DUPLICATES REMOVED)

=> d l3 total ibib kwic


L3   ANSWER 1 OF 19  USPATFULL
ACCESSION NUMBER:        2000:146395  USPATFULL
TITLE:                   Cyclic amine modulators of chemokine receptor activity
INVENTOR(S):             Caldwell, Charles G., Scotch Plains, NJ, United States
                         Maccoss, Malcolm, Freehold, NJ, United States
                         Finke, Paul E., Milltown, NJ, United States
                         Mills, Sander G., Scotch Plains, NJ, United States
                         Oates, Bryan, Wayne, NJ, United States
                         Kothandaraman, Shankaran, Kendall Park, NJ, United
                         States
                         Kim, Dooseop, Westfield, NJ, United States
                         Wang, Liping, Plainsboro, NJ, United States
PATENT ASSIGNEE(S):      Merck & Co., Inc., Rahway, NJ, United States (U.S.
                         corporation)

                              NUMBER         DATE
                         ---------------- --------
PATENT INFORMATION:      US 6140349       20001031
```

APPLICATION INFO.: US 1999-241486 19990201 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1998-73446	19980202 (60)

DOCUMENT TYPE: Utility
PRIMARY EXAMINER: Chang, Ceila
LEGAL REPRESENTATIVE: Thies, J. Eric; Rose, David L.
NUMBER OF CLAIMS: 32
EXEMPLARY CLAIM: 1
LINE COUNT: 3199
SUMM The **peptides** eotaxin, RANTES, MIP-1.alpha., MIP-1.beta., **MCP**-1, and **MCP**-3 are known to bind to **chemokine** receptors. As noted above, the **inhibitors** of HIV-1 replication present in supernatants of CD8+ T cells have been characterized as the .beta.-**chemokines** RANTES, MIP-1.alpha. and MIP-1.beta.. PCT Patent Publication WO 97/10211 and EPO Patent Publication EP 0,673,928 disclose certain piperidines as tachykinin antagonists. PCT Patent Publications WO 97/24325 and WO 97/44329, and Japan Patent Publication JP 09,249,566 disclose certain compounds as **chemokine** antagonists.

L3 ANSWER 2 OF 19 USPATFULL
ACCESSION NUMBER: 2000:142393 USPATFULL
TITLE: Cyclic amine modulations of chemokine receptor activity
INVENTOR(S): Caldwell, Charles G., Scotch Plains, NJ, United States
Finke, Paul E., Milltown, NJ, United States
Maccoss, Malcolm, Freehold, NJ, United States
Meurer, Laura C., Scotch Plains, NJ, United States
Mills, Sander G., Scotch Plains, NJ, United States
Oates, Bryan, Wayne, NJ, United States
PATENT ASSIGNEE(S): Merck & Co., Inc., Rahway, NJ, United States (U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 6136827	20001024
APPLICATION INFO.:	US 1998-120010	19980721 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1997-53754	19970725 (60)

DOCUMENT TYPE: Utility
PRIMARY EXAMINER: Chang, Ceila
LEGAL REPRESENTATIVE: Thies, J. Eric; Rose, David L.
NUMBER OF CLAIMS: 20
EXEMPLARY CLAIM: 1
LINE COUNT: 3161
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
SUMM The **peptides** eotaxin, RANTES, MIP-1.alpha., MIP-1.beta., **MCP**-1, and **MCP**-3 are known to bind to **chemokine** receptors. As noted above, the **inhibitors** of HIV-1 replication present in supernatants of CD8+ T cells have been characterized as the .beta.-**chemokines** RANTES, MIP-1.alpha. and MIP-1.beta.. PCT Patent Publication WO 97/10211 and EPO Patent Publication EP 0,673,928 disclose certain piperidines as tachykinin. .

L3 ANSWER 3 OF 19 USPATFULL
ACCESSION NUMBER: 2000:128351 USPATFULL
TITLE: 3,3-disubstituted piperidines as modulators of chemokine receptor activity
INVENTOR(S): MacCoss, Malcolm, Freehold, NJ, United States
Mills, Sander G., Scotch Plains, NJ, United States
PATENT ASSIGNEE(S): Merck & Co., Inc., Rahway, NJ, United States (U.S.

corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 6124319	20000926
APPLICATION INFO.:	US 1998-9488	19980120 (9)

DOCUMENT TYPE: Utility
PRIMARY EXAMINER: Travers, Russell
LEGAL REPRESENTATIVE: Thies, J. Eric; Rose, David L.
NUMBER OF CLAIMS: 10
EXEMPLARY CLAIM: 1
LINE COUNT: 1901
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
SUMM The **peptides** eotaxin, RANTES, MIP-1-60 , MIP-1.beta., **MCP**-1, and **MCP**-3 are known to bind to **chemokine** receptors. As noted above, the **inhibitors** of HIV-1 replication present in supernatants of CD8+ T cells have been characterized as the .beta.-**chemokines** RANTES, MIP-1.alpha. and MIP-1.beta.. U.S. Pat. Nos. 5,340,822, 5,350,852, 5,434,158, 5,559,132, 5,589,489, and 5,635,510 and PCT Patent Publication WO 95/05377. . .

L3 ANSWER 4 OF 19 USPATFULL
ACCESSION NUMBER: 2000:4806 USPATFULL
TITLE: Spiro-substituted azacycles as modulators of chemokine receptor activity
INVENTOR(S): Mills, Sander G., Scotch Plains, NJ, United States
Maccoss, Malcolm, Freehold, NJ, United States
Springer, Martin S., Westfield, NJ, United States
PATENT ASSIGNEE(S): Merck & Co., Inc., Rahway, NJ, United States (U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 6013644	20000111
APPLICATION INFO.:	US 1997-989940	19971212 (8)

DOCUMENT TYPE: Utility
PRIMARY EXAMINER: Krass, Frederick
LEGAL REPRESENTATIVE: Thies, J. Eric; Rose, David L.
NUMBER OF CLAIMS: 9
EXEMPLARY CLAIM: 1
LINE COUNT: 2845
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
SUMM The **peptides** eotaxin, RANTES, MIP-1.alpha., MIP-1.beta., **MCP**-1, and **MCP**-3 are known to bind to **chemokine** receptors. As noted above, the **inhibitors** of HIV-1 replication present in supernatants of CD8+ T cells have been characterized as the .beta.-**chemokines** RANTES, MIP-1.alpha. and MIP-1.beta.. PCT Patent Publications WO 94/17045 (published Aug. 4, 1994), WO 94/29309 (published Dec. 22, 1994), and. . .

L3 ANSWER 5 OF 19 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2000405934 MEDLINE
DOCUMENT NUMBER: 20305499
TITLE: Angiotensin III increases MCP-1 and activates NF-kappaB and
AP-1 in cultured mesangial and mononuclear cells.
AUTHOR: Ruiz-Ortega M; Lorenzo O; Egido J
CORPORATE SOURCE: Fundacion Jimenez Diaz, Universidad Autonoma, Madrid, Spain.
SOURCE: KIDNEY INTERNATIONAL, (2000 Jun) 57 (6) 2285-98.
Journal code: KVB. ISSN: 0085-2538.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals

ENTRY MONTH: 200010
ENTRY WEEK: 20001004
AB . . . of renal diseases. Angiotensin II (Ang II) participates in inflammatory cell infiltration in the kidney. However, the influence of other **peptides** of the renin-angiotensin system, such as the N-terminal Ang II degradation product Ang III, has not been addressed. METHODS: In. . . cultured renal and mononuclear cells, we investigated whether Ang III is involved in monocyte recruitment through the regulation of the **chemokine**, monocyte chemoattractant protein-1 (**MCP**-1; Northern blot, Western blot, immunofluorescence, and chemotaxis), and the activation of transcription factors, nuclear factor kappaB (NF-kappaB) and activating protein-1 (AP-1; electrophoretic mobility shift assay). RESULTS: In cultured rat mesangial and mononuclear cells, Ang III increased **MCP**-1 gene expression and protein levels. Supernatants from Ang III-treated mesangial cells showed increased chemoattractant activity for monocytes, which was partially inhibited by the addition of anti-**MCP**-1 antibody. Ang III elicited a rapid NF-kappaB activation (8-fold, after 30 min), showing a kinetics and intensity similar to that. . . and disappearance of cytosolic IkappaB. Ang III also activated AP-1 (5-fold, after 18 h), while SP-1 was unchanged. Two NF-kappaB **inhibitors** abolished the Ang III-induced **MCP**-1 mRNA expression, suggesting that overexpression of this **chemokine** is mediated, at least in part, by NF-kappaB activation. CONCLUSIONS: Ang III activates the transcription factors NF-kappaB and AP-1 and increases the expression of related genes, such as **MCP**-1. Our study describes a novel and potent proinflammatory action of this Ang degradation product, expanding the present view of the. . .

L3 ANSWER 6 OF 19 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2000362900 EMBASE
TITLE: Protective effect of Rolipram in experimental autoimmune neuritis: Protection is associated with down-regulation of IFN-.gamma. and inflammatory chemokines as well as up-regulation of IL-4 in peripheral nervous system.
AUTHOR: Abbas N.; Zou L.-P.; Pelidou S.-H.; Winblad B.; Zhu J.
CORPORATE SOURCE: J. Zhu, Division of Geriatric Medicine (B84), Karolinska Institute, Huddinge Hospital, S-141 86 Huddinge, Sweden. Jie.Zhu@neurotec.ki.se
SOURCE: Autoimmunity, (2000) 32/2 (93-99).
Refs: 26
ISSN: 0891-6934 CODEN: AUIMEI
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 030 Pharmacology
037 Drug Literature Index
008 Neurology and Neurosurgery
026 Immunology, Serology and Transplantation
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Rolipram, a phosphodiesterase type 4 **inhibitor**, is reported to have anti-inflammatory effects. It can markedly downregulate antigen-driven T cell proliferation and suppress TNF-.alpha. and TNF-.beta. production. . . IFN-.gamma. and TNF-.alpha. production. Here we report that EAN induced in Lewis rats by inoculation with the PNS P2 protein **peptide** 57-81 and Freund's complete adjuvant (FCA), was strongly suppressed by Rolipram administered twice daily intraperitoneally from day 9 post immunization. . . of clinical EAN to day 18 p.i. This clinical effect was associated with dose-dependent down-regulated production of IFN-.gamma. and the **chemokines** macrophage inflammatory protein-1.alpha. (MIP-1.alpha.), MIP-2 and monocyte chemotactic protein-1 (**MCP**-1) as well as up-regulated IL-4

production in sciatic nerve sections from Rolipram-treated EAN rats at maximum of clinical EAN, i.e.. . .

L3 ANSWER 7 OF 19 USPATFULL

ACCESSION NUMBER: 1999:163409 USPATFULL

TITLE: Functional expression of mammalian adenylyl cyclase in yeast

INVENTOR(S): Broach, James R., Princeton, NJ, United States
Manfredi, John P., Ossining, NY, United States
Trueheart, Joshua, Nyack, NY, United States

PATENT ASSIGNEE(S): Cadus Pharmaceutical Corporation, Tarrytown, NY, United States (U.S. corporation)

	NUMBER	DATE	
PATENT INFORMATION:	US 6001553	19991214	
	WO 9530012	19951109	
APPLICATION INFO.:	US 1997-732218	19970114	(8)
	WO 1995-US5149	19950426	
		19970114	PCT 371 date
		19970114	PCT 102(e) date

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1994-233700, filed on 26 Apr 1994, now abandoned

DOCUMENT TYPE: Utility
PRIMARY EXAMINER: Wax, Robert A.
LEGAL REPRESENTATIVE: Lahive & Cockfield LLP; DeConti, Jr., Giulio A.; Lauro, Peter C.
NUMBER OF CLAIMS: 83
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Figure(s); 3 Drawing Page(s)
LINE COUNT: 4954
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD The present invention may be used to identify **inhibitors** or activators of many mammalian receptors, including but not limited to, receptor tyrosine kinases and cytokine receptors (such as those for IL-3, IL-5, GM-CSF, M-CSF and EPO etc.), G protein-coupled **chemokine** receptors (such as RANTES, **MCP**-3, **MCP**-1, MIP-1.alpha. and IL-8 receptor), growth factor receptors (such as EGFR and PDGFR etc.), and multi-subunit immune recognition receptors also known. . . as MIRRs (such as Fc.epsilon.RI, and Fc.gamma.RII etc.). Further receptors useful in the current invention include the G protein-coupled C5a **peptide** receptor, the thrombin **peptide** receptor (PAR1) and its homolog PAR2, formyl **peptide** and bradykinin receptors, muscarinic receptors, adrenergic receptors, melatonin, galanin, glucagon and orphan receptors and transporter proteins such as the multidrug. . .

L3 ANSWER 8 OF 19 USPATFULL

ACCESSION NUMBER: 1999:121364 USPATFULL

TITLE: Spiro-substituted azacycles as modulators of chemokine receptor activity

INVENTOR(S): Mills, Sander G., Scotch Plains, NJ, United States
Maccoss, Malcolm, Freehold, NJ, United States
Springer, Martin S., Westfield, NJ, United States

PATENT ASSIGNEE(S): Merck & Co., Inc., Rahway, NJ, United States (U.S. corporation)

	NUMBER	DATE	
PATENT INFORMATION:	US 5962462	19991005	
APPLICATION INFO.:	US 1997-989947	19971212	(8)

NUMBER	DATE

PRIORITY INFORMATION: US 1996-32735 19961213 (60)
US 1996-33558 19961220 (60)

DOCUMENT TYPE: Utility
PRIMARY EXAMINER: Dees, Jose G.
ASSISTANT EXAMINER: Oazi, Sabiha N.
LEGAL REPRESENTATIVE: Thies, J. Eric; Rose, David L.
NUMBER OF CLAIMS: 10
EXEMPLARY CLAIM: 1
LINE COUNT: 6786
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
SUMM The **peptides** eotaxin, RANTES, MIP-1.alpha., MIP-1.beta., **MCP**-1, and **MCP**-3 are known to bind to **chemokine** receptors. As noted above, the **inhibitors** of HIV-1 replication present in supernatants of CD8+ T cells have been characterized as the .beta.-**chemokines** RANTES, MIP-1.alpha. and MIP-1.beta.. PCT Patent Publications WO 94/17045 (published Aug. 4, 1994), WO 94/29309 (published Dec. 22, 1994), and. . .

L3 ANSWER 9 OF 19 USPATFULL
ACCESSION NUMBER: 1999:75632 USPATFULL
TITLE: Substituted aminoquinolines as modulators of chemokine receptor activity
INVENTOR(S): Hagmann, William K., Westfield, NJ, United States
Springer, Martin S., Westfield, NJ, United States
PATENT ASSIGNEE(S): Merck & Co., Inc., Rahway, NJ, United States (U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 5919776	19990706
APPLICATION INFO.:	US 1997-993494	19971218 (8)

DOCUMENT TYPE: Utility
PRIMARY EXAMINER: Mach, D. Margaret M.
LEGAL REPRESENTATIVE: Thies, J. Eric; Rose, David L.
NUMBER OF CLAIMS: 12
EXEMPLARY CLAIM: 1
LINE COUNT: 1808
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
SUMM The **peptides** eotaxin, RANTES, MIP-1.alpha., MIP-1.beta., **MCP**-1, and **MCP**-3 are known to bind to **chemokine** receptors. As noted above, the **inhibitors** of HIV-1 replication present in supernatants of CD8+ T cells have been characterized as the .beta.-**chemokines** RANTES, MIP-1.alpha. and MIP-1.beta.. Certain substituted aminoquinoline derivatives have been described as **inhibitors** of C5a receptor binding (Lanza, et al., J. Med. Chem., 35, 252-258 (1992)).

DUPLICATE 2

L3 ANSWER 10 OF 19 MEDLINE
ACCESSION NUMBER: 1999272411 MEDLINE
DOCUMENT NUMBER: 99272411
TITLE: Monocyte arrest and transmigration on inflamed endothelium in shear flow is inhibited by adenovirus-mediated gene transfer of IkappaB-alpha.
AUTHOR: Weber K S; Draude G; Erl W; de Martin R; Weber C
CORPORATE SOURCE: Institut fur Prophylaxe und Epidemiologie der Kreislaufkrankheiten, Ludwig-Maximilians Universitat, Munchen, Germany.. kim.weber@klp.med.uni-muenchen.de
SOURCE: BLOOD, (1999 Jun 1) 93 (11) 3685-93.
Journal code: A8G. ISSN: 0006-4971.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals; Cancer Journals
ENTRY MONTH: 199908
ENTRY WEEK: 19990804

AB Mobilization of nuclear factor-kappaB (NF-kappaB) activates transcription of genes encoding endothelial adhesion molecules and **chemokines** that contribute to monocyte infiltration critical in atherogenesis. Inhibition of NF-kappaB has been achieved by pharmacological and genetic approaches; however, monocyte interactions with activated endothelium in shear flow following gene transfer of the NF-kappaB **inhibitor** IkappaB-alpha have not been studied. We found that overexpression of IkappaB-alpha in endothelial cells using a recombinant adenovirus prevented tumor. . . molecule-1 (VCAM-1), intercellular adhesion molecule-1 (ICAM-1), and E-selectin mRNA and surface protein expression and the upregulation of transcripts for the **chemokines** monocyte chemoattractant protein 1 (**MCP**-1) and growth-related activity-alpha (GRO-alpha) by TNF-alpha. This was associated with a reduction in endothelial **MCP**-1 secretion and GRO-alpha immobilization. Adhesion assays under physiological shear flow conditions showed that firm arrest, spreading, and transmigration of monocytes on TNF-alpha-activated endothelium was markedly inhibited by IkappaB-alpha overexpression. Inhibition with monoclonal antibodies and **peptide** antagonists inferred that this was due to reduced expression of Ig integrin ligand as well as of **chemokines** specifically involved in these events. In contrast, rolling of monocytes was increased by IkappaB-alpha transfer and was partly mediated by. . .

L3 ANSWER 11 OF 19 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 1999289324 MEDLINE
DOCUMENT NUMBER: 99289324
TITLE: Identification of oligopeptide sequences which inhibit migration induced by a wide range of chemokines.
AUTHOR: Reckless J; Grainger D J
CORPORATE SOURCE: Department of Medicine, University of Cambridge, Addenbrookes Hospital, Box 157, Hills Road, Cambridge CB2 2QQ, UK.. jr219@mole.bio.cam.ac.uk
SOURCE: BIOCHEMICAL JOURNAL, (1999 Jun 15) 340 (Pt 3) 803-11. Journal code: 9YO. ISSN: 0264-6021.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
ENTRY MONTH: 199910
ENTRY WEEK: 19991001
AB We have identified an amino acid sequence, termed **peptide** 3, corresponding to amino acids 51-62 of the mature human monocyte chemoattractant protein-1 (**MCP**-1), which inhibits human mononuclear-cell and THP-1-cell migration induced by a wide range of **chemokines**. For example, **peptide** 3 inhibited **MCP** -1-induced THP-1 migration in a transwell assay with an ED50 of approx. 8 microM. **Peptide** 3 binds directly to THP-1 cells with an association constant of approx. 10 microM, and is therefore likely to be a direct receptor antagonist for CC and CXC **chemokine** receptors. By performing a structure-function analysis of this **peptide**, we have identified a sequence variant that shows an approx. 3-4-fold greater potency as an **inhibitor** of **chemokine**-induced migration [Leu4Ile11 **peptide** 3 (1-12)]. Furthermore, unlike **peptide** 3, which binds to the Duffy antigen receptor for **chemokines** on human erythrocytes with a similar affinity to the specific **chemokine** receptors on THP-1 cells, the Leu4Ile11 **peptide** 3 (1-12) sequence variant shows at least 20-fold greater selectivity for the specific receptors. Derivatives of Leu4Ile11 **peptide** 3 (1-12) are therefore the best candidates among the molecules we have investigated for use as a **chemokine** **inhibitor** in vivo.

L3 ANSWER 12 OF 19 USPATFULL
ACCESSION NUMBER: 1998:33576 USPATFULL
TITLE: Hematopoietic cells: compositions and methods

INVENTOR(S): Taichman, Russell S., Ann Arbor, MI, United States
Emerson, Stephen G., Wayne, PA, United States
PATENT ASSIGNEE(S): The Regent of the University of Michigan, Ann Arbor, MI, United States (U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 5733541	19980331
APPLICATION INFO.:	US 1995-426792	19950421 (8)

DOCUMENT TYPE: Utility
PRIMARY EXAMINER: Chambers, Jasemine C.
ASSISTANT EXAMINER: Clark, Deborah J. R.
LEGAL REPRESENTATIVE: Arnold, White & Durkee
NUMBER OF CLAIMS: 41
EXEMPLARY CLAIM: 1
LINE COUNT: 3768
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
SUMM TABLE 3

CYTOKINES

Angiogenin
Chemokines
C10
Epithelial Neuturophil Activating **Peptide**-78 (ENA-78)
Growth Related (GRO)-.alpha.
GRO-.alpha.
GRO-.beta.
GRO-gamma
Macrophage Inhibitory Protein-1 (MIP-1)
MIP-1.alpha.
MIP-1.beta.
Monocyte Chemoatractant Protein-1 2 and 3 (**MCP**)
Regulated upon Activation Normal T-cell Expressed and Secreted (RANTES)
Interleukin-8 (IL-8)
Epidermal Growth Factors (EGF)
Amphiregulin (AR)
Beta-Cellulin
Epidermal Growth Factor (EGF)
Heparin Binding-EGF
TGF-.alpha.
Fibroblast Growth Factors (aFGF)
Acidic FIbroblast. . . (No other Name that I am aware of)
Insulin Like Growth Factors-I and II (IGF-I and II)
Interferons (IFN)
IFN-Alpha
IFN-Beta
IFN-Gamma
Interleukins
Interleukin-1 alpha
Interleukin-1 beta
Interleukin-2
Interleukin-3
Interleukin-5
Interleukin-6
Interleukin-7
Interleukin-8
Interleukin-9
Interleukin-10
Interleukin-11
Interleukin-12
Interleukin-13
Latency Associated **Peptide** (LAP)
Leukemia Inhibitory Factor (LIF)
Macrophage Colony Stimulating Factor (M-CSF)

.beta.-Nerve
Oncostatin-M (OSM)
Osteoclast-Colony Stimulating Factor
Platelet Derived Growth Factors
Alpha & Beta Heterodimers and Homodimers (PDGF)
Pleiokine Family
Pleiotrophin
Midkine
Secretory Leukocyte Protease **Inhibitor**
Stem Cell Factor (SCF or c-Kit Ligand)
Transforming Growth Factor Beta Factors (TGF-Beta)
TGF-.beta..sub.1 Through TGF-.beta..sub.2
Thromobopoietin
Tumor Necrosis Factors (TNF's)
TNF-.alpha.
TNF-.beta. (Lymphotoxin)
Vascular Endothelial Growth Factor
VEGF
Placenta. . .

L3 ANSWER 13 OF 19 MEDLINE DUPLICATE 4

ACCESSION NUMBER: 1999003150 MEDLINE
DOCUMENT NUMBER: 99003150
TITLE: Helicobacter pylori lipopolysaccharide binds to CD14 and stimulates release of interleukin-8, epithelial neutrophil-activating peptide 78, and monocyte chemotactic protein 1 by human monocytes.
AUTHOR: Bliss C M Jr; Golenbock D T; Keates S; Linevsky J K; Kelly C P
CORPORATE SOURCE: Section of Gastroenterology, Boston Medical Center, Boston University School of Medicine, Boston, Massachusetts 02118, USA.
CONTRACT NUMBER: DK54920 (NIDDK)
DK02128 (NIDDK)
GM54060 (NIGMS)
SOURCE: INFECTION AND IMMUNITY, (1998 Nov) 66 (11) 5357-63.
Journal code: GO7. ISSN: 0019-9567.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
ENTRY MONTH: 199901
ENTRY WEEK: 19990104

AB . . . by leukocyte infiltration of the gastric mucosa. The aims of this study were to determine whether H. pylori-derived factors stimulate **chemokine** release from human monocytes and to ascertain whether H. pylori lipopolysaccharide (LPS) may be responsible for this effect. Human peripheral. . . blood monocytes were exposed to an H. pylori water extract (HPE) or to purified H. pylori LPS. Levels of the **chemokines** interleukin-8 (IL-8), epithelial neutrophil-activating **peptide** 78 (ENA-78), and monocyte chemotactic protein 1 (**MCP**-1) were measured by enzyme-linked immunosorbent assay. The contribution of H. pylori LPS to monocyte activation was determined by using the. . . sphaeroides lipid A (RSLA) and a blocking monoclonal antibody to CD14 (60bca). HPE increased monocyte secretion of IL-8, ENA-78, and **MCP**-1. Heat treatment of HPE did not reduce its ability to activate monocytes. Purified H. pylori LPS also stimulated monocyte **chemokine** production but was 1,000-fold less potent than Salmonella minnesota lipid A. RSLA blocked H. pylori LPS-induced monocyte IL-8 release in. . . (by 88%, $P < 0.01$), whereas the nonblocking anti-CD14 monoclonal antibody did not. These experiments with potent and specific LPS **inhibitors** indicate that the main monocyte-stimulating factor in HPE is LPS. H. pylori LPS, acting through CD14, stimulates human monocytes to release the neutrophil-activating

chemokines IL-8 and ENA-78 and the monocyte-activating **chemokine MCP**-1. Despite its low relative potency, H. pylori LPS may play an important role in the pathogenesis of H. pylori gastritis.

L3 ANSWER 14 OF 19 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 1998129353 MEDLINE
DOCUMENT NUMBER: 98129353
TITLE: RANTES activation of phospholipase D in Jurkat T cells: requirement of GTP-binding proteins ARF and RhoA.
AUTHOR: Bacon K B; Schall T J; Dairaghi D J
CORPORATE SOURCE: Department of Immunobiology, DNAX Research Institute, Palo Alto, CA 94304, USA.. Kbacon@neurocrine.com
SOURCE: JOURNAL OF IMMUNOLOGY, (1998 Feb 15) 160 (4) 1894-900. Journal code: IFB. ISSN: 0022-1767.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals; Cancer Journals
ENTRY MONTH: 199804
AB The **chemokine** RANTES is a potent agonist of T cell activation. In an investigation of signal-transduction events activated by this **chemokine**, we have shown that RANTES stimulates dose-dependent phospholipase D (PLD) activity in Jurkat cells. Equilibrium-binding analyses using 125I-labeled RANTES indicated. . . approximately 600 sites per cell, and a binding specificity that was not comparable with that of any of the known **chemokine** receptors, since 125I-labeled RANTES was displaced by macrophage-inflammatory protein-1 beta (but not macrophage-inflammatory protein-1 alpha), monocyte-chemotactic protein-1 (**MCP**-1), **MCP**-3, **MCP**-4, and eotaxin. RANTES-induced PLD activation was augmented by GTP gamma S, but not GDP beta S, and inhibited by the protein kinase C **inhibitor** bisindolylmaleimide, as well as the fungal metabolite brefeldin A, and C3 exoenzyme (Clostridium botulinum), implicating the activation of RhoA. RANTES. . . immunoprecipitated RhoA. RANTES-stimulated PLD activity was dependent on an ADP-ribosylation factor(s), as assessed by inhibition studies using a synthetic inhibitory **peptide** of the N-terminal 16 amino acids of ADP-ribosylation factor 1. These studies indicate the potential existence of a novel receptor-mediated mechanism for activation of T cells by the **chemokine** RANTES.

L3 ANSWER 15 OF 19 MEDLINE
ACCESSION NUMBER: 97272158 MEDLINE
DOCUMENT NUMBER: 97272158
TITLE: Human glomerular mesangial cell phagocytosis of apoptotic neutrophils: mediation by a novel CD36-independent vitronectin receptor/thrombospondin recognition mechanism that is uncoupled from chemokine secretion.
AUTHOR: Hughes J; Liu Y; Van Damme J; Savill J
CORPORATE SOURCE: Department of Medicine, University Hospital, Nottingham, United Kingdom.. jeremy.hughes@nottingham.ac.uk
SOURCE: JOURNAL OF IMMUNOLOGY, (1997 May 1) 158 (9) 4389-97. Journal code: IFB. ISSN: 0022-1767.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals; Cancer Journals
ENTRY MONTH: 199707
AB . . . from inflamed glomeruli, thereby promoting resolution of glomerulonephritis. Mesangial cell phagocytosis of apoptotic neutrophils in vitro was not affected by **inhibitors** of lectin-like receptors, phosphatidylserine receptors, the 61D3 Ag, and beta1 and beta2

integrins, receptors which have been implicated in phagocytosis. . . apoptotic cells by particular populations of semiprofessional and professional phagocytes. However, the specific inhibitory effects of cationic aminosugars, Arg-Gly-Asp-Ser (RGDS) **peptide**, and mAbs to phagocyte alpha(v)beta3 vitronectin receptor integrin and "bridging" thrombospondin 1 (TSP1) indicated that mesangial cell phagocytosis of apoptotic. . . and sulfatides. Nevertheless, phagocytosis of apoptotic neutrophils by either mesangial cells or Mphi failed to elicit secretion of IL-8 and **MCP**-1, representatives of each major class of proinflammatory chemotactic cytokines. We conclude that mesangial cell phagocytosis of apoptotic neutrophils involves a novel CD36-independent, alpha(v)beta3/TSP-mediated mechanism that is uncoupled from **chemokine** secretion, emphasizing the injury-limiting potential of mesangial cell phagocytosis of apoptotic cells.

L3 ANSWER 16 OF 19 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 97101427 MEDLINE
DOCUMENT NUMBER: 97101427
TITLE: Endogenous modulators of TNF and IL-1 response are under partial control of TNF in baboon bacteremia.
AUTHOR: Redl H; Schlag G; Paul E; Bahrami S; Buurman W A; Strieter R M; Kunkel S L; Davies J; Foulkes R
CORPORATE SOURCE: Ludwig Boltzmann Institute for Experimental and Clinical Traumatology, Vienna, Austria.
SOURCE: AMERICAN JOURNAL OF PHYSIOLOGY, (1996 Nov) 271 (5 Pt 2) R1193-8.
Journal code: 3U8. ISSN: 0002-9513.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199703
ENTRY WEEK: 19970302
AB Tumor necrosis factor (TNF) and interleukin (IL)-1 are two cytokines for which naturally occurring **inhibitors** have been identified. The present study was undertaken to evaluate the extent to which scavenging of TNF in bacteremia attenuates. . . and TNF and was significantly attenuated by anti-TNF treatment, as were the circulating levels of IL-1, IL-8, and monocyte chemotactic **peptide**-1 (**MCP**-1) in the anti-TNF Ab group. We conclude that the increase in circulating natural cytokine modulators observed in nonhuman primate bacteremia. . . of endogenous TNF because it was influenced by anti-TNF pretreatment. This attenuation is comparable to the anti-TNF effect on the **chemokine** **MCP**-1.

L3 ANSWER 17 OF 19 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 96003442 MEDLINE
DOCUMENT NUMBER: 96003442
TITLE: Serum amyloid A induces calcium mobilization and chemotaxis of human monocytes by activating a pertussis toxin-sensitive signaling pathway.
AUTHOR: Badolato R; Johnston J A; Wang J M; McVicar D; Xu L L; Oppenheim J J; Kelvin D J
CORPORATE SOURCE: Biologic Carcinogenesis and Development Program, Program Resources, Inc./Dyncorp, Frederick, MD, USA.
SOURCE: JOURNAL OF IMMUNOLOGY, (1995 Oct 15) 155 (8) 4004-10.
Journal code: IFB. ISSN: 0022-1767.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals; Cancer Journals

ENTRY MONTH: 199601
AB . . . mechanism of SAA signaling is unknown, we have investigated the possibility that SAA, like other chemoattractants such as the chemotactic **peptide** FMLP and **chemokines**, might induce migration of monocytes by G protein activation. We report here that preincubation of monocytes with pertussis toxin (PTx) inhibited SAA chemotaxis, while incubation with cholera toxin (CTx) did not. Staurosporine and H-7, both **inhibitors** of protein kinase C (PKC), significantly decreased rSAA-induced chemotaxis of monocytes, suggesting that PKC may be involved in the rSAA. . . by rSAA, was comparable to that elicited by FMLP, and was severalfold greater than that induced by optimal concentrations of **chemokine** beta-family members such as RANTES, MCAF/**MCP** -1, and MIP-1 alpha. The chemoattractants FMLP, RANTES, MIP-1 alpha, and MCAF/**MCP**-1, all failed to desensitize rSAA-induced Ca2+ influx and chemotaxis in monocytes. This suggests that SAA uses a distinct receptor that. . .

DUPLICATE 8

L3 ANSWER 18 OF 19 MEDLINE
ACCESSION NUMBER: 96163236 MEDLINE
DOCUMENT NUMBER: 96163236
TITLE: Interleukin-1-induced IL-8 and IL-6 gene expression and production in human mesangial cells is differentially regulated by cAMP.
AUTHOR: Robson R L; Westwick J; Brown Z
CORPORATE SOURCE: Department of Pharmacology, University of Bath, Avon, England, United Kingdom.
SOURCE: KIDNEY INTERNATIONAL, (1995 Dec) 48 (6) 1767-77.
Journal code: KVB. ISSN: 0085-2538.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199605
AB . . . the initiation and propagation of inflammatory events within the glomerulus via the generation of the mesangioproliferative cytokine IL-6 and the **chemokines** IL-8 and **MCP**-1. The objective of this study was to investigate the role of cAMP in the regulation of IL-6 and IL-8 gene expression and **peptide** production in IL-1 stimulated human MC. Agents known to elevate cAMP, including dibutyryl cAMP (db-cAMP), forskolin or isobutyl-methylxanthine (IBMX) were. . . the presence of IL-1, all three agents produced a marked potentiation of IL-6 mRNA expression and dose-dependent increase in IL-6 **peptide** production (twofold), but had little or no effect on IL-8 mRNA expression or **peptide** generation. In marked contrast cholera toxin (CT) caused a dose-dependent potentiation of both IL-1-induced IL-6 (approximately fourfold) and IL-8 **peptide** (approximately twofold) generation. The control agent, the purified binding subunit of cholera toxin (CT-B) which is devoid of ADP-ribosylating activity also enhanced IL-6 and IL-8 (approximately twofold) **peptide** generation indicating cAMP-independent mechanisms may be involved in the CT up-regulation of these cytokines. Treatment of MC with the cycloxygenase **inhibitor** indomethacin resulted in partial inhibition (37%) of IL-6 production but had no effect on IL-8 generation. Thus our data show. . .

DUPLICATE 9

L3 ANSWER 19 OF 19 MEDLINE
ACCESSION NUMBER: 96003901 MEDLINE
DOCUMENT NUMBER: 96003901
TITLE: The protein phosphatase inhibitor calyculin A stimulates chemokine production by human synovial cells.
AUTHOR: Jordan N J; Watson M L; Westwick J
CORPORATE SOURCE: Department of Pharmacology, University of Bath, Claverton Down, U.K.
SOURCE: BIOCHEMICAL JOURNAL, (1995 Oct 1) 311 (Pt 1) 89-95.
Journal code: 9YO. ISSN: 0264-6021.
PUB. COUNTRY: ENGLAND: United Kingdom

Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
ENTRY MONTH: 199601
AB Cultured human synovial fibroblasts express mRNA for the chemotactic cytokines (**chemokines**) interleukin-8 (IL-8), monocyte chemotactic protein 1 (**MCP**-1) and regulated upon activation normal T-cell expressed and presumably secreted (RANTES), when stimulated with IL-1 or tumour necrosis factor alpha (TNF alpha). Calyculin A, a potent type 1/2A protein serine/threonine phosphatase **inhibitor**, was used to examine the role of protein phosphatases in the regulation of **chemokine** gene expression. Calyculin A (1 nM) mimicked IL-1 by inducing IL-8 and **MCP**-1 mRNA expression in synovial cells. IL-8 mRNA was induced over a similar time period (1-6 h) in response to IL-1 or calyculin A, whereas **MCP**-1 mRNA was induced more rapidly (1-2 h) by calyculin A than by IL-1 (4-6 h). Expression of RANTES mRNA occurred. . . of protein phosphatase type 1/2A may have a differential role in the regulation of the expression of each of the **chemokine** genes. Synovial fibroblasts also secreted IL-8 and IL-6 **peptide** when stimulated with either IL-1/TNF alpha or calyculin A. The amount of IL-8 and IL-6 **peptide** produced in response to calyculin A was significantly increased above that produced by untreated synovial cells, though it was much. . . acted synergistically with IL-1 or TNF alpha to cause a 2-fold potentiation of IL-1- or TNF alpha-induced IL-8 mRNA and **peptide** and RANTES mRNA expression. These results suggest that although inhibition of a protein phosphatase may be able to regulate the magnitude of IL-1-induced **chemokine** gene expression, the IL-1 signal transduction pathway involves components in addition to phosphatase inhibition, possibly including the activation of a. . .

=> s chemokine (s) peptide (s) inhibitor (s) mcp (s) treatment

L4 10 CHEMOKINE (S) PEPTIDE (S) INHIBITOR (S) MCP (S) TREATMENT

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PROCESSING COMPLETED FOR L4
L5 4 DUP REM L4 (6 DUPLICATES REMOVED)

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L5 ANSWER 1 OF 4 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2000362900 EMBASE
TITLE: Protective effect of Rolipram in experimental autoimmune neuritis: Protection is associated with down-regulation of IFN-.gamma. and inflammatory chemokines as well as up-regulation of IL-4 in peripheral nervous system.
AUTHOR: Abbas N.; Zou L.-P.; Pelidou S.-H.; Winblad B.; Zhu J.
CORPORATE SOURCE: J. Zhu, Division of Geriatric Medicine (B84), Karolinska Institute, Huddinge Hospital, S-141 86 Huddinge, Sweden. Jie.Zhu@neurotec.ki.se
SOURCE: Autoimmunity, (2000) 32/2 (93-99).
Refs: 26
ISSN: 0891-6934 CODEN: AUIMEI
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 030 Pharmacology
037 Drug Literature Index
008 Neurology and Neurosurgery
026 Immunology, Serology and Transplantation

LANGUAGE: English
SUMMARY LANGUAGE: English
AB Rolipram, a phosphodiesterase type 4 **inhibitor**, is reported to have anti-inflammatory effects. It can markedly downregulate antigen-driven T cell proliferation and suppress TNF-.alpha. and TNF-.beta. production in vitro and in vivo, which have led to its use in the **treatment** of a number of autoimmune disorders including experimental autoimmune encephalomyelitis (EAE) and experimental autoimmune neuritis (EAN). EAN is a CD4+. . . IFN-.gamma. and TNF-.alpha. production. Here we report that EAN induced in Lewis rats by inoculation with the PNS P2 protein **peptide** 57-81 and Freund's complete adjuvant (FCA), was strongly suppressed by Rolipram administered twice daily intraperitoneally from day 9 post immunization. . . of clinical EAN to day 18 p.i. This clinical effect was associated with dose-dependent down-regulated production of IFN-.gamma. and the **chemokines** macrophage inflammatory protein-1.alpha. (MIP-1.alpha.), MIP-2 and monocyte chemotactic protein-1 (**MCP**-1) as well as up-regulated IL-4 production in sciatic nerve sections from Rolipram-treated EAN rats at maximum of clinical EAN, i.e.. . . cell-dependent autoimmune diseases and inflammatory neuropathies. These observations call for further studies on the potential role of Rolipram in the **treatment** of autoimmune diseases.

L5 ANSWER 2 OF 4 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 1999003150 MEDLINE
DOCUMENT NUMBER: 99003150
TITLE: Helicobacter pylori lipopolysaccharide binds to CD14 and stimulates release of interleukin-8, epithelial neutrophil-activating peptide 78, and monocyte chemotactic protein 1 by human monocytes.
AUTHOR: Bliss C M Jr; Golenbock D T; Keates S; Linevsky J K; Kelly C P
CORPORATE SOURCE: Section of Gastroenterology, Boston Medical Center, Boston University School of Medicine, Boston, Massachusetts 02118, USA.
CONTRACT NUMBER: DK54920 (NIDDK)
DK02128 (NIDDK)
GM54060 (NIGMS)
SOURCE: INFECTION AND IMMUNITY, (1998 Nov) 66 (11) 5357-63.
Journal code: GO7. ISSN: 0019-9567.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
ENTRY MONTH: 199901
ENTRY WEEK: 19990104
AB . . . by leukocyte infiltration of the gastric mucosa. The aims of this study were to determine whether H. pylori-derived factors stimulate **chemokine** release from human monocytes and to ascertain whether H. pylori lipopolysaccharide (LPS) may be responsible for this effect. Human peripheral. . . blood monocytes were exposed to an H. pylori water extract (HPE) or to purified H. pylori LPS. Levels of the **chemokines** interleukin-8 (IL-8), epithelial neutrophil-activating **peptide** 78 (ENA-78), and monocyte chemotactic protein 1 (**MCP**-1) were measured by enzyme-linked immunosorbent assay. The contribution of H. pylori LPS to monocyte activation was determined by using the. . . sphaeroides lipid A (RSLA) and a blocking monoclonal antibody to CD14 (60bca). HPE increased monocyte secretion of IL-8, ENA-78, and **MCP**-1. Heat **treatment** of HPE did not reduce its ability to activate monocytes. Purified H. pylori LPS also stimulated monocyte **chemokine** production but was 1,000-fold less potent than Salmonella minnesota lipid A. RSLA blocked H. pylori LPS-induced monocyte IL-8 release in. . . (by 88%, $P < 0.01$), whereas

the nonblocking anti-CD14 monoclonal antibody did not. These experiments with potent and specific LPS **inhibitors** indicate that the main monocyte-stimulating factor in HPE is LPS. H. pylori LPS, acting through CD14, stimulates human monocytes to release the neutrophil-activating **chemokines** IL-8 and ENA-78 and the monocyte-activating **chemokine MCP**-1. Despite its low relative potency, H. pylori LPS may play an important role in the pathogenesis of H. pylori gastritis.

L5 ANSWER 3 OF 4 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 97101427 MEDLINE
DOCUMENT NUMBER: 97101427
TITLE: Endogenous modulators of TNF and IL-1 response are under partial control of TNF in baboon bacteremia.
AUTHOR: Redl H; Schlag G; Paul E; Bahrami S; Buurman W A; Strieter R M; Kunkel S L; Davies J; Foulkes R
CORPORATE SOURCE: Ludwig Boltzmann Institute for Experimental and Clinical Traumatology, Vienna, Austria.
SOURCE: AMERICAN JOURNAL OF PHYSIOLOGY, (1996 Nov) 271 (5 Pt 2) R1193-8.
Journal code: 3U8. ISSN: 0002-9513.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199703
ENTRY WEEK: 19970302
AB Tumor necrosis factor (TNF) and interleukin (IL)-1 are two cytokines for which naturally occurring **inhibitors** have been identified. The present study was undertaken to evaluate the extent to which scavenging of TNF in bacteremia attenuates. . . 10(9) colony-forming units/kg live Escherichia coli over 2 h and were subjected to either placebo or anti-TNF antibody (anti-TNF Ab) **treatment** (1 mg/kg CDP571, Celltech, UK) 2 h before E. coli infusion (observation time: 72h). IL-1ra (range: 50-100 ng/ml) and sTNFR. . . 75 kDa, 30-35 ng/ml) release was more sustained than that of IL-1 and TNF and was significantly attenuated by anti-TNF **treatment**, as were the circulating levels of IL-1, IL-8, and monocyte chemotactic **peptide**-1 (**MCP**-1) in the anti-TNF Ab group. We conclude that the increase in circulating natural cytokine modulators observed in nonhuman primate bacteremia. . . of endogenous TNF because it was influenced by anti-TNF pretreatment. This attenuation is comparable to the anti-TNF effect on the **chemokine MCP**-1.

L5 ANSWER 4 OF 4 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 96163236 MEDLINE
DOCUMENT NUMBER: 96163236
TITLE: Interleukin-1-induced IL-8 and IL-6 gene expression and production in human mesangial cells is differentially regulated by cAMP.
AUTHOR: Robson R L; Westwick J; Brown Z
CORPORATE SOURCE: Department of Pharmacology, University of Bath, Avon, England, United Kingdom.
SOURCE: KIDNEY INTERNATIONAL, (1995 Dec) 48 (6) 1767-77.
Journal code: KVB. ISSN: 0085-2538.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199605
AB . . . the initiation and propagation of inflammatory events within the glomerulus via the generation of the mesangioproliferative cytokine IL-6 and the **chemokines** IL-8 and **MCP**-1. The objective of

this study was to investigate the role of cAMP in the regulation of IL-6 and IL-8 gene expression and **peptide** production in IL-1 stimulated human MC. Agents known to elevate cAMP, including dibutyryl cAMP (db-cAMP), forskolin or isobutyl-methylxanthine (IBMX) were. . . the presence of IL-1, all three agents produced a marked potentiation of IL-6 mRNA expression and dose-dependent increase in IL-6 **peptide** production (twofold), but had little or no effect on IL-8 mRNA expression or **peptide** generation. In marked contrast cholera toxin (CT) caused a dose-dependent potentiation of both IL-1-induced IL-6 (approximately fourfold) and IL-8 **peptide** (approximately twofold) generation. The control agent, the purified binding subunit of cholera toxin (CT-B) which is devoid of ADP-ribosylating activity also enhanced IL-6 and IL-8 (approximately twofold) **peptide** generation indicating cAMP-independent mechanisms may be involved in the CT up-regulation of these cytokines. **Treatment** of MC with the cycloxygenase **inhibitor** indomethacin resulted in partial inhibition (37%) of IL-6 production but had no effect on IL-8 generation. Thus our data show. . .

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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	33.36	33.57

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